SCL & GP Complications and Their Management

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Causes of CL Complications

- Physical, lens fitting
- CL BVP
- CL dehydration
- Deposits/wettability
- Altered blinking
- Hypoxia
- Mechanical damage

- Pre-existing systemic or ocular disease
- Environmental factors
- Chemical/toxic/allergic
- Microbiological
- Immunological
- Non-compliance
Causes of CL Complications

*If CL Related-

Physiological
- Mechanical
- Toxic/Allergic
- Hypoxia
- Inflammatory
- Microbial/Infectious
Corneal Epithelial Staining

- Dehydration
- Toxic
- Exposure
- Mechanical
- Hypoxia
SMILE

- Prevalence
  - up to 25% of SCL wearing subjects have inferior corneal staining (Guillon et al, 1990)
  - majority of severe staining is inferiorly positioned (Schwallie et al, 1997)

- Etiology
  - initial lens dehydration
  - depletion of the postlens tear film
  - subsequent staining through epithelial desiccation
  - greatest with
    - high water content lenses
    - thinner lenses
    - low humidity environments
    - incomplete blinking
SMILE

- Symptoms
  - dryness
  - reduced wearing time

- Signs
  - coarse inferior punctate staining in an arcuate fashion
  - extends from 4 to 8 o'clock in the lower 1/3 of the cornea
  - typically 4-5 mm in from the limbus
SMILE

- **Management**
  - **Minor**
    - no treatment
  - **Major**
    - ocular lubricants
    - blinking exercises
    - locally placed humidifiers
    - switch to
      - low water content lenses
      - thicker higher water content lenses

- **Prognosis**
  - variable
  - inter patient differences
Corneal Epithelial Staining

- Dehydration
- Toxic
- Exposure
- Mechanical
- Hypoxia
Toxic/Allergic

- Sensitivity to preservatives (CL-SLK)
- Toxicity to traces of peroxide, high concentrations of preservatives (SPK or pseudodendrites)
- Sensitivity to preservative plus bound deposit (CLAPC)
Toxicity Staining

- **Incidence**
  - unknown; small percentage compared with THI & CHX-preserved care regimens
  - occurs with all preservatives (delayed)
  - occasionally with enzyme removers
  - acute with non-neutralized peroxide

- **Etiology**
  - toxic or hypersensitivity reaction to component in care system

- **Symptoms**
  - occasionally asymptomatic
  - reduced wearing time
  - dryness with lenses
  - stinging on lens insertion
Toxicity Staining

- Signs
  - bulbar/limbal conjunctival hyperaemia
  - diffuse corneal SPK
  - palpebral hyperemia
Solution Sensitivity Management

- Discontinue lens wear temporarily
- Change lens solutions
  - (especially the preservative)
Lens Associated Superior Limbic Kerato-Conjunctivitis: Signs

- Typically bilateral
- Superior bulbar and limbal hyperaemia
- Apron of redundant folds of bulbar conjunctiva at superior limbus
- Conjunctival chemosis
- Infiltrates (grey)
- Sub-epithelial haze
- SCL wearer
Lens Associated Superior Limbic Kerato-Conjunctivitis: Signs

- Corneal and conjunctival staining
  - fluorescein/Rose Bengal

- Limbal hypertrophy

- Palpebral response
  - papillae/redness

- Signs remain well after cessation of wear

- Differential diagnosis - SLK of Theodore
CL-SLK Management

- Distinguish from SLK of Theodore
- Discontinue lens wear
- Monitor recovery
- Lubrication
- Change lens design/lens fit
Management

- Fit RGP lenses (probably the best option)
- Use alternative solutions
  - Preservative–free
  - Alternate preservative
- Steroid therapy
CONTACT LENS PAPILLARY CONJUNCTIVITIS

SIGNS

- Enlarged papillae
- Roughened appearance
  (irregular specular reflection)
- Palpebral redness

Tissue oedema

Precursor to CL-Associated Papillary Conjunctivitis
CLAPC
CONTACT LENS PAPILLARY CONJUNCTIVITIS

AETIOLOGY

- Lens front surface deposits
  - mechanical irritation
  - immune response
  - drying of lens surface
CONTACT LENS PAPILLARY CONJUNCTIVITIS MANAGEMENT

- Modify lens wear
- Change lens design
- Frequent lens replacement
- Optimize lens care and maintenance
CONTACT LENS PAPILLARY CONJUNCTIVITIS

MANAGEMENT

• RGP lenses
• Pharmacological therapy
• Patient education
• Complete resolution is unlikely
Corneal Epithelial Staining

- Dehydration
- Toxic
- Exposure
- Mechanical
- Hypoxia
Mechanical or Physical Factors

- Lens movement, fitting relationship i.e. exposure staining
- Lens rigidity, i.e. SEAL
- Physical condition of lens i.e. tears, scratches, deposits i.e. CLAPC
- Blinking: completeness, lagophthalmos, lid tonus
SEAL

- **Prevalence**
  - 8% of SCL wearers (Hine et al, 1987)
  - lower with current lens types?

- **Etiology**
  - multifactorial
    - mechanical, hypoxia and dehydration all implicated
  - mechanical trauma from inflexible lens designs
  - misalignment between lens and ocular surface at limbus
  - pressure induced by the top lid produces staining
    - (Young & Mirejovsky, 1993)
SEAL

○ Symptoms
  ● often none
  ● occasionally FB sensation
  ● occasionally mild irritation upon lens removal

○ Signs
  ● arcuate staining 1mm from the superior limbus
  ● between 10 o'clock and 2 o'clock.
  ● staining runs parallel to the limbus
  ● 0.1- 0.3mm wide and 2-5mm in length
SEAL

- Management
  - remove lenses and cease wear for 3-4 days
    - resolves in > 30% of cases
  - fit a different BOZR of the same design
  - fit a thinner, more flexible lens material
  - fit a lens with a thinner periphery
  - if problem persists refit with a rigid lens
    - problem with HDK lenses
Corneal Epithelial Staining

- Dehydration
- Toxic
- Exposure
- Mechanical
- Hypoxia
Hypoxic

Corneal Edema: **Chronic Responses**

**Epithelial**
- Microcysts
- Bullae (Bullous Keratopathy)
- Edematous Corneal Formations (ECF)
- Microcystic Edema

**Stromal**
- Stromal Thinning
- Neovascularisation

**Endothelial**
- Polymegethism
- Endothelial Bedewing
- Guttatae
Dk/t Required: NO Edema

- **Daily Wear**
  - 20.0 (Sarver et al, 1981)
  - 24.1 (Holden & Mertz, 1984)
  - Harvitt & Bonanno, 1999
    - basal epithelial cells: 23.0
    - entire corneal thickness: 35.0

- **Extended Wear**
  - 75.0 (O’Neal et al, 1984)
  - 87.0 (Holden & Mertz, 1984)
  - Harvitt & Bonanno, 1999
    - basal epithelial cells: 89.0
    - entire corneal thickness: 125.0
Stromal Striae

- **Etiology**
  - hypoxia
  - accumulation of lactic acid in cornea
  - osmotic shift
  - fluid enters the cornea
  - occurs with > 5% corneal edema
  - number increases with increasing edema

- **Symptoms**
  - none

- **Signs**
  - fine vertically orientated lines in the posterior stroma
Stromal Striae

### Table: Striae and Folds

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<th>Number</th>
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- **Management**
  - increase corneal oxygenation

- **Prognosis**
  - excellent

*La Hood & Grant, 1990*
STROMAL STRIAE
MANAGEMENT

- Intervention is warranted
- Increase lens Dk/t
- Reduce wearing time
CORNEAL OEDEMA
MANAGEMENT

- Minimize contact lens effects:
  - maximize lens Dk/t (priority)
  - optimize lens fit (less significant)
  - fit siloxane hydrogels

- Decrease lens wear
ENDOTHELIAL FOLDS
MANAGEMENT

- Intervention is mandatory
- Significant increase in lens Dk\(t\) needed
- Very short wearing time
- Refit with siloxane hydrogels, or high Dk RGP lenses
Limbal Hyperemia

- **Prevalence**
  - 20-50% clinically significant (Pritchard et al, 1996)
  - very common in all lens types
    - greatest with SCL
    - 100% to some degree?

- **Etiology**
  - hypoxia
    - hyperemia related to Dk/t (Papas et al, 1995)
  - mechanical irritation
  - tight lens
Limbal Hyperemia

○ Signs
  ● engorgement of limbal blood vessels
  ● linked to vascularisation?

○ Management
  ● optimise lens fit
  ● reduce wear time
  ● increase lens transmissibility
    ○ new high Dk SCL materials
Limbal Hyperemia

Dumbleton et al, 1998

Time (months)

Mean Limbal Hyperemia

p=0.000
Endothelial Blebs

- **Prevalence**
  - 100% of neophyte wearers
  - not in silicone lenses
  - reduced in adapted wearers

- **Etiology**
  - Endothelial cell edema
    - acidic pH shift (stromal acidosis)
      - increase in carbonic acid (hypercapnia)
      - increase in lactic acid (hypoxia)
  - Bulging of endothelial cells produces an optically empty area

Bonnano & Polse, 1987
The diagram illustrates the structure of a cell, specifically highlighting a 'blebbbed' cell. The cell is depicted with a yellowish color, and the surrounding tissue is marked as endothelium and stroma. The diagram uses arrows to indicate the direction of processes or interactions within or around the cell.
Endothelial Blebs

- **Symptoms**
  - None

- **Signs**
  - Black, non-reflecting areas within the endothelium
  - Appear as holes
    - within 10 mins of insertion
    - peak after 20-30 mins
    - subside after 2-3 hours

Zantos & Holden, 1977
Clinical significance is unclear

Higher lens Dk/t minimizes:

- the number of blebs
- the magnitude of the response
Unreversed effect

Diverging refractor (low RI; fluid vacuole)

Reversed effect

Converging refractor (high RI; microcyst)

Zantos, 1983
Compromised Epithelium

- Anoxia
  - Suboptimal metabolism
    - Slowed mitosis
  - Microcysts
    - 15 - 50 μm inclusions in the epithelium
    - Sign of chronic hypoxia
    - Appear as scattered dots
    - Take 2-3 months to occur
    - 4-6 months to peak
  - Vacuoles
    - Fluid-filled bubbles
stroma

intra-epithelial sheet

microcysts

microcyst breaking through surface (stains with fluorescein)

growth

epithelium

stroma

Zantos, 1981
Microcysts

Marginal retro illumination
Microcysts
EPITHELIAL MICROCYSTS

AETIOLOGY: EFFECT OF Dk/t

Keay et al., 2000
EPITHELIAL MICROCYSTIS

MANAGEMENT

• Careful monitoring
  - occur in non-lens wearers as well
• If < 10, no action is needed
• Increasing number warrants intervention
  - increase lens DK/t
  - reduce wearing time
• Rebound effect after lens discontinuation
• Lengthy time to resolve
EPITHELIAL MICROCYSTS
AETIOLOGY: ‘REBOUND’

Keay et al., 2000

Extended wear

Microcysts

Low Dk/t lens (6N EW) → High Dk/t lens (30N EW)

Pre-Lens

1M 3M 6M 9M 12M 1M 3M

Extended wear

Rebound

Change

Keay et al., 2000
Microcysts

- **Management**
  - < 30 microcysts
    - no action required
    - monitor carefully
  - > 30 microcysts
    - cease lens wear (1 month)
    - change from EW to DW
    - refit with HDK lenses

- **Prognosis**
  - initial increase in number
  - slow decrease thereafter
  - disappear after 3 months
    - *(Holden et al, 1985)*
Vascularisation

- **Prevalence**
  - 11% of all wearers (Keech et al, 1996)
    - < 1% of RGP (Levy, 1985)
    - < 5% of DW SCL (Poggio & Abelsen, 1993)
    - < 15% of EW SCL (Spoor et al, 1984)

- **Etiology**
  - hypoxia produces stromal edema and softening
  - vasostimulating agent produces vessel growth
    - epithelial damage
    - solution toxicity
    - infection
Vascularisation

- Symptoms
  - none

- Signs
  - new vessels in the cornea
    - superficial
    - deep stromal (very rare)
Vascularisation

- **Management**
  - If ‘mild’
    - increase lens transmissibility
    - reduce wearing time
    - monitor carefully
  - If ‘severe’
    - refit with high Dk RGP
    - cease wear permanently
    - refit with high Dk SCL?
Vascularisation

Mean Grade of Neovascularization

Time (months)

LDK

HDK

Dumbleton et al, 1998
Vascularisation

- Prognosis
  - vessels empty rapidly
  - ghost vessels remain (years?)
  - ghost vessel can refill
CORNEAL VASCULARIZATION

MANAGEMENT

• Change lens solutions
• Patient education and follow-up
• Optimize fitting characteristics
• Decrease wearing time
• Refit with silicone hydrogel or RGP lenses
Polymegethism

- **Prevalence**
  - natural process of ageing
  - CL accelerate the process
  - greatest with low Dk lenses (Schoessler, 1983; Schoessler et al, 1984)

- **Etiology**
  - chronic hypoxia produces tissue-acidosis (Bonanno & Polse, 1987)
  - acidic shift at endothelium
    - hypoxia: lactic acid
    - hypercapnia: carbonic acid
  - cell fluid balance disturbed
  - possible structural damage
Polymegethism

- **Symptoms**
  - usually none
  - may result in "corneal exhaustion syndrome"? (Sweeney, 1992)

- **Signs**
  - ratio of smallest:largest endothelial cells increases
    - from 1:5 to 1:20
  - endothelium includes cells of significantly differing sizes
Polymegethism

- **Management**
  - not reversible
  - if suggestive of long-term hypoxia
    - alleviate tissue acidosis
    - refit with high transmissibility lenses

- **Prognosis**
  - poor
  - very slow recovery (if any)
ENDOTHELIAL POLYMEGATHISM

MANAGEMENT

• Preventative strategy
  - high Dk/t lenses
• Careful assessment with slit lamp
• Increase lens oxygen transmissibility
• Minimal recovery expected
Inflammatory Responses

- **AI (asymptomatic infiltrates)**
  - Single, anywhere, in anterior stroma, no staining or anterior chamber reaction or redness

- **IK (infiltrative keratitis)**
  - Peripheral, multiple, slight staining and redness, no anterior chamber reaction, can be bilateral

- **AIK (asymptomatic infiltrative keratitis)**
  - Peripheral, small, focal, punctate staining, mild to moderate redness, no anterior chamber reaction, can be bilateral
Inflammation

- Toxic, CL-SLK
- Allergic, SEI
- Sensitivity to Gm –ve endotoxins, CLARE
- Sensitivity to Staph (+ve) exotoxins, CLPU
- Mechanical stimulus: CL-denatured protein (CLAPC) or adherence of micro-organisms as PA or SA, SEAL?
Inflammation

○ Inflammation vs Infection?
  • clinical diagnosis
“Presumed” Sterile

- inflammatory response
- peripheral / mid-peripheral lesions
- lesions 1-2 mm
- circular appearance
- mild pain
- epithelium - intact or staining
- mild epiphora
- mild to moderate injection
- confined to anterior stroma only
- mild (if any) corneal suppuration
- minimal AC reaction

Stapleton et al, 1993
Grant et al, 1998
“Presumed” Microbial

- infective process
- paracentral / central lesions
- lesions > 1mm
- irregular appearance
- pain increasing; may be severe
- epithelial defect
- intense epiphora
- moderate to severe injection
- anterior to mid-stromal
- severe, progressive corneal suppuration
- ac flare and occasional hypopyon

Stapleton et al, 1993
Grant et al, 1998
Sterile Infiltrates

- **Prevalence**
  - 1% of non-lens wearers (Sweeney et al, 1996)
    - higher risk in smokers
  - 2-10% of lens wearers (Gorden & Kracher, 1985; Cutter et al, 1996)
    - higher with SCL, particularly EW SCL (Josephson & Caffery, 1979; Vajdic et al, 1995; Cutter et al, 1996)

- **Etiology**
  - inflammatory cells migrate from limbal vessels
    - PMN
  - inflammatory response to numerous factors
    - bacteria; closed-eye environment; tight lens
    - hypoxia; lens deposits; care systems
Sterile Infiltrates

- **Signs**
  - circular areas of haziness
  - normally in the limbal area
  - focal or diffuse
  - conjunctival hyperemia
Appearance
Infiltrative Keratitis

- Symptomatic or asymptomatic
- DW, EW & no wear
- With or without epithelial staining
- Predilection for 4 and 8 o’clock position
- Staph exotoxins?
CORNEAL INFILTRATES: SUMMARY

Anterior Stromal Infiltrate

- **Peripheral/anywhere**
  - Very small, focal
  - Single and/or diffuse infiltration

- **Peripheral**
  - Small, focal
  - Several, and/or mild to moderate diffuse infiltration

- **Peripheral/mid-peripheral**
  - Mild-moderate diffuse infiltration and/or small, focal infiltrate, several possible

**AI**
- Very small, focal
- Single and/or diffuse infiltration

**AIK**
- Small, focal
- Several, and/or mild to moderate diffuse infiltration

**IK**
- Very small, focal
- Single and/or diffuse infiltration

**Macrophage**

**Neutrophil (PMN)**

**Lymphocyte**

Anterior Stromal Infiltrate
Contact Lens Associated Red Eye (CLARE)

- EW only
- Moderately painful eye
- Significant generalised hyperaemia & epiphora
  - typically at night
- Multiple limbal infiltrates
  - no staining
- Rapid resolution
- Associated with gram –ve bacteria

Holden et al, 1996
Sankaridurg et al, 1996
CLARE MANAGEMENT

- Temporary discontinuation of wear
- Palliative therapy
  - saline rinse (sterile)
  - lubrication
- Regular lens replacement
- Low toxicity lens care products
CLARE MANAGEMENT

- Monitor recovery
  - complete resolution of infiltrates
  - about 1-3 weeks
- Re-start with DW initially
- Caution with continued EW
  - optimize fitting
  - change lens type
  - change lens care products
- Monitor for recurrence
Contact Lens Peripheral Ulcer (CLPU)

- SCL EW only
- Focal, circumscribed, round infiltrate
  - 0.1 - 1.5mm diameter
- Full thickness loss of epithelium, but Bowmans is intact
  - epithelial staining
  - leaves scar
- Predilection for under the lid
- Due to toxins from staph or other gram +ve colonising lens?

Willcox et al., 1995
Holden et al., 1999
CONTACT LENS PERIPHERAL ULCER

MANAGEMENT

• Discontinue lens wear immediately
• Generally, healing is rapid
• Monitor carefully for first 24 hrs
• Prophylaxis
• Resolves with scarring
Inflammation - Management

- Clear wearer instruction
- Eliminate possibility of infection
- Remove lens
- Refer if necessary/unsure

Treatment
- change to DW
- lid hygiene measures
- avoid wearing EWSCL when unwell
Accompanying staining requires intervention

- lens wear discontinuation
- monitoring
- antibiotic prophylaxis?

Resolution (clear cornea) necessary before lens wear continued
CORNEAL INFILTRATES
MANAGEMENT

Risk of recurrence
Need to reduce risk
• isolate cause
• change lenses, solutions, care routine
• change wear schedule
• patient re-education

Refit with daily disposables, siloxane hydrogels, or GPs
Use preservative free products
Microbial/Infectious

- Bacterial Ulcers
- Fungal Ulcers
- Protozoan Ulcers
- Viral Infections
- Microbial Keratitis
Signs & Symptoms Can Be Assessed Using:

- Biomicroscope
- Keratometer/Videokeratoscope
- Retinoscope/Refraction
- Case History
Keratometry

Clinical Signs:
- Lens Flexure (RGP)
- Lens Warpage (RGP)
- Corneal Distortion (SCL)
  - Related to edema
  - Thick lenses
  - Rigidity of materials
Retinoscopy

Clinical Signs:
- Poor Optics
- Poor Surface Wettability
- Power Change
- Lens Deposits
- Uncorrected Astigmatism
- Toric Lens Rotation
- BV? Early presbyopia?
- Reproducibility, accuracy?
Case History

Symptoms:
- CL- Induced Dry Eye
- Discomfort
  - Lens Related
  - Eye Related
  - Solution Related
- Irritation
GP Fitting Complications
“3&9” Staining

Prevalence

- 50-80% of daily wear RGP lens wearers
  - (Lowther, 1982; Barr, 1985)
- 10% clinically significant (Ghormley et al, 1990)
“3&9” Staining

- **Aetiology**
  - Drying of the peripheral cornea in association with an unstable tear film
  - Causes are multifactorial
    - poor peripheral lens fit
    - inadequate blinking
    - poor lens wettability
    - abnormal tear composition
“3&9” Staining

- Symptoms
  - Dry, gritty, irritable eyes and reduced wearing time

- Signs
  - Conjunctival hyperaemia along the horizontal meridian in conjunction with epithelial punctate staining at the four and eight o'clock positions
  - Extreme cases may develop a pseudopterygium, vascularised limbal keratitis or dellen
“3&9” Staining Management

- If insignificant corneal astigmatism exists
  - increase the diameter, reduce the edge clearance, reduce the thickness profile and reduce the edge thickness
- If significant corneal astigmatism exists
  - fit a fully back surface toric with a large overall diameter and minimal edge clearance
- All cases may benefit from blinking exercises and artificial lubricants
- In severe cases the final resort may be to refit with a soft lens
RGP Lens Binding

- **Prevalence**
  - more prevalent in RGP EW
    - 50% on eye opening *(Swarbrick & Holden, 1989)*
    - as high as 80% *(Zabkiewicz et al, 1987; Lin et al, 1989)*
  - can occur with DW RGP
RGP Lens Binding

- **Etiology**
  1. proposed patient related factors
     - corneal astigmatism; eyelid pressure; corneal thickness;
     - ocular rigidity; peripheral topography; tear film characteristics
  2. proposed lens related factors
     - lens diameter; peripheral curve design; specific gravity
     - edge clearance; flexibility; lens thickness

- **most likely** *(Swarbrick, 1988)*
  - lens is pushed down onto the cornea at night by the upper lid
  - post lens tear film is eliminated
  - highly viscous mucous layer acts as an adhesive
  - lens is literally "glued" to the cornea
RGP Lens Binding

- **Symptoms**
  - none
  - possible complaints re lens removal (difficult)
  - occasional spectacle blur following removal

- **Signs**
  - immobile, decentred lens
  - indentation ring remains when the lens is removed
RGP Lens Binding

**Management**
- increase lens movement
  - smaller
  - increased edge clearance
  - flatter or steeper BOZR?
- change to daily wear RGP's
- change to FRP RGP's *(Woods & Efron, 1996)*
- refit with soft lenses

**Prognosis**
- variable
- inter-patient differences
High Riding RGP
High Riding Lenses

- **Patient factors**
  - tight lids
  - displaced corneal apex
  - high WRT corneal cyl

- **Lens factors**
  - centrally too flat
  - too large
  - too much axial edge clearance
  - too thick mid-periphery
    - could be due to being high minus lens
High Riding Lenses

- Patient factors
  - tight lids
    - refit with smaller/bigger lens
    - refit with SCL
  - displaced corneal apex
    - refit with bigger RGP
    - refit with SCL
  - high WRT corneal cyl
    - refit with smaller spherical RGP
    - refit with bigger BS toric RGP
High Riding Lenses

- **Lens factors**
  - centrally too flat
    - Steepen BOZR
  - too large
    - refit with smaller diameter RGP
  - too much axial edge clearance
    - reduce AEL of lens
  - too thick mid-periphery
    - lenticulate to reduce thickness profile
    - reduce centre thickness?
Dimple Veil

- **Prevalence**
  - unknown
  - predominantly rigid lens wearers
  - very rarely SCL wearers

- **Etiology**
  - not “true” staining
  - trapped air bubbles
  - poor fitting relationship between cornea and lens
  - usually observed in
  - steep central fitting lenses
  - high riding lenses in cases of high WTR astigmatism
Dimple Veil

- **Symptoms**
  - none

- **Signs**
  - small indentations in the corneal epithelium
  - appearance similar to the surface of a golf ball
Dimple Veil

- **Management**
  - fit a flatter BOZR
  - reduce the edge clearance
  - reduce the overall size
  - control the thickness profile
    - to reduce the high riding position
  - change to a toric back surface lens
    - with a highly astigmatic cornea

- **Prognosis**
  - excellent
The End!